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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER
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KAUSHAL, SUMESH

ART UNIT	PAPER NUMBER
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1636

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DATE MAILED: 06/03/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

<b>Office Action Summary</b>	<b>Application No.</b>	<b>Applicant(s)</b>
	09/696,867	BRUNKOW ET AL.
	<b>Examiner</b> Sumesh Kaushal Ph.D.	<b>Art Unit</b> 1636

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

## Status

1)  Responsive to communication(s) filed on 20 March 2003 .

2a)  This action is **FINAL**.                    2b)  This action is non-final.

3)  Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

## Disposition of Claims

4)  Claim(s) 34-45 is/are pending in the application.

4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.

5)  Claim(s) \_\_\_\_\_ is/are allowed.

6)  Claim(s) 34-45 is/are rejected.

7)  Claim(s) \_\_\_\_\_ is/are objected to.

8)  Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

## Application Papers

9)  The specification is objected to by the Examiner.

10)  The drawing(s) filed on 10/25/00 is/are: a)  accepted or b)  objected to by the Examiner. \* See 870-948  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)  The proposed drawing correction filed on \_\_\_\_\_ is: a)  approved b)  disapproved by the Examiner.  
If approved, corrected drawings are required in reply to this Office action.

12)  The oath or declaration is objected to by the Examiner.

**Priority under 35 U.S.C. §§ 119 and 120**

13)  Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)  All b)  Some \* c)  None of:

1.  Certified copies of the priority documents have been received.
2.  Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
3.  Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

14)  Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a)  The translation of the foreign language provisional application has been received.

15)  Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

**Attachment(s)**

1)  Notice of References Cited (PTO-892)      4)  Interview Summary (PTO-413) Paper No(s). \_\_\_\_ .  
2)  Notice of Draftsperson's Patent Drawing Review (PTO-948) ~~✓~~      5)  Notice of Informal Patent Application (PTO-152)  
3)  Information Disclosure Statement(s) (PTO-1449) Paper No(s) 13,14 .      6)  Other: \_\_\_\_\_

## **DETAILED ACTION**

Applicant's response filed on 03/20/03 has been acknowledged.

### ***Continued Examination Under 37 CFR 1.114***

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 03/20/03 has been entered.

*Claims 43-45 are newly filed.*

*Claims 34-42 are amended.*

*Claims 34-45 are pending and are examined in this office action.*

► *Applicants are advised to follow Amendment Practice under revised 37 CFR §1.121 (<http://www.uspto.gov/web/offices/pac/dapp/ola/preognocice/revamdtprac.htm>). Each amendment document that includes a change to an existing claim, or submission of a new claim, must include a complete listing of all claims in the application. After each claim number, the status must be indicated in a parenthetical expression, and the text of each claim under examination (with markings to show current changes) must be presented. The listing will serve to replace all prior versions of the claims in the application.*

### ***Claim Rejections - 35 USC § 112***

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

1. Claims 36-45 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter, which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The scope of invention as claimed (after the recent amendment) encompasses a transgenic mouse or a transgenic non-human mammal (rat, rabbit, sheep, goat or pig), whose cells express an  $Fkh^{sf}$  transgene comprising nucleic acid molecule comprising a sequence at least 90% identical to the coding region of SEQ ID NO:1 or 3, wherein the expression of such  $Fkh^{sf}$  transgene results in the reduction of T-lymphocyte proliferation in the mammal (as claimed).

At best the specification discloses only disclosed that the nucleic acid sequences of SEQ ID NO:1 encoding the amino acid sequences of SEQ ID NO:2 is mutant form of mouse  $Fkh^{sf}$  gene, the expression of which results in the lymphoproliferative disorder in a transgenic mouse (page 32, example-1). The specification fails to disclose that the nucleic acid sequences of SEQ ID NO:2 encoding the amino acid sequences of SEQ ID NO:4 is a mutant form of human  $Fkh^{sf}$  gene, the expression of which results in the lymphoproliferative disorder in a transgenic mouse. The scope of invention as claimed encompasses substitution, addition and/or deletion of at least 10% (90% identical) of nucleic acid sequences in the nucleic acid of SSEQ ID NO:1 and 3. The specification fails to disclose any variant of SEQ ID NO:1 or 3 explicitly or implicitly that modulates reduction of T-lymphocyte proliferation in any mammal.

Applicant is referred to the Interim guidelines on Written Description published December 21, 1999 in the Federal Register, Vol. 64, No. 244, pp. 71427-71440. The disclosure

of a single species is rarely, if ever, sufficient to describe a broad genus, particularly when the specification fails to describe the features of that genus, even in passing. (see *In re Shokal* 113USPQ283(CCPA1957); *Purdue Pharma L. P. vs Faulding Inc.* 56 USPQ2nd 1481 (CAFC 2000). In the instant case the specification only teaches human Fkh<sup>sf</sup> and mouse Fkh<sup>sf</sup> nucleic acid sequences of SEQ ID NO: 1 and 3 respectively but fails to disclose any variant of SEQ ID NO:1 or 3 that has the functional property of Fkh<sup>sf</sup> polypeptide explicitly or implicitly as putatively considered by the applicant.

The possession may be shown by actual reduction to practice, clear depiction of the invention in a detailed drawing, or by describing the invention with sufficient relevant identifying characteristics (as it relates to the claimed invention as a whole) such that a person skilled in the art would recognize that the inventor had possession of the claimed invention. *See, e.g., Pfaff v. Wells Electronics, Inc.*, 525 U.S. 55, 68, 119 S.Ct. 304, 312, 48 USPQ2d 1641, 1647 (1998); *Eli Lilly*, 119 F.3d at 1568, 43 USPQ2d at 1406; *Amgen, Inc. v. Chugai Pharmaceutical*, 927 F.2d 1200, 1206, 18 USPQ2d 1016, 1021 (Fed. Cir. 1991). In claims to genetic material, generic statement such as "vertebrate insulin cDNA" or mammalian insulin cDNA," without more, is not adequate written description of claimed genus, since it does not distinguish genus from others except by function, and does not specifically define any of genes that fall within its definition, or describe structural features commonly possessed by members of genus that distinguish them from others; accordingly, naming type of material generally known to exist, in absence of knowledge as to what that material consists of, is not description of that material (*Eli Lilly*, 119 F.3d at 1568, 43 USPQ2d at 1406).

In the instant case the nucleic acid variants (as claimed) has been defined only by a statement of function that broadly encompasses reduction of T-lymphocyte proliferation in a non-human mammal like mouse, rat, rabbit, sheep, goat and pig, which conveyed no distinguishing information about the identity of the claimed DNA sequence, such as its relevant structural or physical characteristics. The variation as claimed also encompasses the conserved motifs, which are considered germane to the functional activity of Fkh<sup>sf</sup> polypeptide. Furthermore 10% variation (90% identical) as claimed would certainly affect proper folding and biological activity if amino acids that are critical for such functions are substituted, since the relationship between the sequence of a polypeptide and its tertiary structure is neither well understood nor predictable. Furthermore, mere identification of critical regions would not be sufficient, as the ordinary artisan would immediately recognize that the encoded polypeptide must assume the proper three-dimensional configuration to be active, which is dependent upon the surrounding residues (see Ngo, in The Protein Folding Problem and Tertiary Structure Prediction, Merz et al. (eds.), Birkhauser Boston: Boston, MA, pp. 433 and 492-495, 1994). Rudinger (in Peptide Hormones, Parsons (ed.), University Park Press: Baltimore, MD, pp. 1-7, 1976). According to these facts, one skill in the art would conclude that applicant was not in the possession of the claimed genus because a description of only one member of this genus is not representative of the variants of genus and is insufficient to support the claim.

2. Claim 34-45 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a transgenic *Scurfy mouse* whose somatic and germ cells express a

transgene comprising a 30kb fragment of normal genomic DNA, including ~7kb coding region of  $Fkh^{sf}$  gene as well as ~20kb of upstream flanking sequence and ~4kb of down stream sequences that contain a sequence encoding mouse  $Fkh^{sf}$  protein wherein the expression of exogenous  $Fkh^{sf}$  transgene results in reduction of T-lymphocyte proliferation in the *scurfy mouse*, does not reasonably provide enablement for any transgenic mouse, rat, rabbit, sheep goat or pig whose cells express an  $Fkh^{sf}$  transgene encoding mouse  $Fkh^{sf}$  (SEQ ID NO:1) or human  $Fkh^{sf}$  (SEQ ID NO:3), wherein the expression of the  $Fkh^{sf}$  transgene results in reduction of T-lymphocyte proliferation in the mammal. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

**Nature Of Invention:**

Invention relates to a transgenic *Scurfy mouse* (*mut.  $Fkh^{sf}$  gene*) wherein the expression of a  $Fkh^{sf}$ -transgene comprising a normal  $Fkh^{sf}$  gene results in reduction of T-lymphocyte proliferation in the mammal.

**Breadth Of Claims And Guidance Provided By The Inventor:**

The scope of invention as claimed encompasses transgenic mouse, rat, rabbit, sheep goat or pig whose cells express an  $Fkh^{sf}$  transgene encoding mouse  $Fkh^{sf}$  (SEQ ID NO:2), wherein the expression of the  $Fkh^{sf}$  transgene results in reduction of T-lymphocyte proliferation in the mammal. The scope of invention as claimed encompasses introduction of a  $Fkh^{sf}$  transgene comprising  $Fkh^{sf}$ -mouse or  $Fkh^{sf}$ -human nucleotide sequences into non-human mammals like mouse, rat, rabbit, sheep goat or pig. In addition the scope of invention as claimed encompasses a transgenic mouse, rat, rabbit, sheep goat or pig whose cells express an  $Fkh^{sf}$  transgene

encoding any and all variants of mouse  $Fkh^{sf}$  (SEQ ID NO:1) or any and all variants of human  $Fkh^{sf}$  (SEQ ID NO:3), wherein the expression of the  $Fkh^{sf}$  transgene (*a  $Fkh^{sf}$  variant*) results in reduction of T-lymphocyte proliferation in the mammal.

The specification teaches isolation of mouse (SEQ ID NO:1) and human (SEQ ID NO:3)  $Fkh^{sf}$  DNA sequences (spec. page 32, example-1). The specification teaches that a two base pair insertion in normal  $Fkh^{sf}$  transgene resulted in *Scurfy phenotype* in mice (page 32, line 20-25). At best the specification only teaches the generation of transgenic mouse (*Scurfy mouse*) wherein a 30 kb fragment of the normal genomic DNA, including the ~7 kb coding region of the  $Fkh^{sf}$  gene, as well as ~20 kb of upstream flanking sequences and ~4 kb of downstream sequences (FIG. 5) was microinjected into mouse one-cell embryos. Five individual founder animals were generated (spec. page 33, lines 9-17). The specification further disclosed that analysis of *sf* (*Scurfy*) progeny reviled that the expression of  $Fkh^{sf}$  transgene in *sf* mice over come the lymphoproliferative defect found in scurfy mice. The specification concluded that addition of the normal  $Fkh^{sf}$  gene can overcome the defect found in scurfy mice, confirming that the mutation in the  $Fkh^{sf}$  gene is the cause of *Scurfy disease* (spec. page 33, lines 25-27, figures 6-8).

Besides a transgenic mouse (*Scurfy mouse*) specification fails to disclose any other mammal like rat, rabbit, sheep goat or pig whose cells express an  $Fkh^{sf}$  transgene encoding mouse  $Fkh^{sf}$  (SEQ ID NO:2) or human  $Fkh^{sf}$  (SEQ ID NO:4), wherein the expression of the  $Fkh^{sf}$  transgene results in reduction of T-lymphocyte proliferation in the mammal. Considering the instant disclosure it is even unclearly whether a mutation in normal  $Fkh^{sf}$  gene would result in *Scurfy disease* in rat, rabbit, sheep goat or pig. The specification even fails to disclose a *Scurfy* model for rat, rabbit, sheep goat or pig. On the other hand the specification fails to disclose that

mere over expression of *normal*  $Fkh^{sf}$  gene (mouse  $Fkh^{sf}$  or human  $Fkh^{sf}$ ) would result in the reduction of T-lymphocyte proliferation in any normal (*non-scruffy*) transgenic mammal as claimed.

**State Of Art And Predictability:**

The state of transgenic art at the time of filing was such that phenotype of an animal is determined by a complex interaction of genetics and environment. (Wood. Comp. Med. 50(1): 12-15, 2000, see page 12; *ref. of record*). The phenotype examined in a transgenic and knock out model is influenced by genetic background, which is the collection of all genes present in an organism that influence a trait or traits. The genes may be part of same biochemical or signaling pathway or of an opposing pathway or may appear unrelated to the gene being studied. Furthermore, allelic variations and the interactions between the allelic variants also influence a particular phenotype. These epigenetic effects can dramatically alter the observed phenotype and therefore can influence or alter the conclusions drawn from the transgenic or knockout models (Sigmund, Arterioscler. Throm. Vasc. Biol. 20:1425-1429, 2000, see page 1425; *ref. of record*).

The transgene expression and physiological consequences of transgene products in non-mouse mammals are not always accurately predicted among various species of mammals (Wall RJ Theriogenology 45:57-68, 1996; *ref. of record*). Transgene efficiency is low, and range from 1% in farm animals (cattle, sheep, pigs) to 3% in laboratory animals like rabbits, mice and rats (Wall, see page 61). Furthermore, the lack of understanding of essential genetic control elements make it difficult to predict the behavior of a transgene in any and all animals because the expression is influenced by position effect in transgenic animals. The individual gene of

interest, promoter, enhancer, coding or non-coding sequences present in the transgene construct and the site of integration, are the important factors that govern the expression of a transgene (Wall, page 61-62). The cis acting elements of one species may interact with different transactivating factors in other species. For example, the introduction of human growth hormone transgene in mice results in mammoth mouse phenotype, whereas expression of the same transgene in pig results in premature death of transgenic pigs. (Pursel VG et al J. Reprod Fert. Sup 40: 235-245 1990, see page 235, para.1; *ref. of record*).

Furthermore, many biochemical pathways are plastic in nature, which reflects the ability of the embryo to use alternative gene when the preferred gene is modified. It is known in the art that the level and the specificity of a transgene as well as the phenotype of the transgenic animal are greatly dependent upon the specific expression vector used. The individual gene of interest, promoter, enhancer, coding or non-coding sequences present in the transgene construct and the site of integration, for example are the important factors that govern the expression of a transgene. (Kappel et al. Current Opinion in Biotechnology 3:558-553 1992; see page 550, col.1, para. 3-4, page 548, col.2 para.2; *ref. of record*).

Furthermore, the phenotype of targeted mutations by a homologous recombination have not always been as predicted because the homologous recombination is a rare event which requires numerous steps that often fail. The embryonic stem (ES) cells are very sensitive to culture conditions and have natural tendency to differentiate, giving rise to unstable genome. The homologous recombination is a rare event in ES cells and the injection of ES in the blastocyte is highly unpredictable (Viville, in Transgenic Animals, Houdebine (eds), Harwood academic publishers, France. pp307-321, 1997; *ref. of record*).

In addition, it is general knowledge in the art that even conservative amino acid substitutions can adversely affect proper folding and biological activity if amino acids that are critical for such functions are substituted, and the relationship between the sequence of a polypeptide and its tertiary structure is neither well understood nor predictable. The mere identification of critical regions would not be sufficient, as the ordinary artisan would immediately recognize that the encoded polypeptide must assume the proper three-dimensional configuration to be active, which is dependent upon the surrounding residues. see Ngo, in The Protein Folding Problem and Tertiary Structure Prediction, Merz et al. (eds.), Birkhauser Boston: Boston, MA, pp. 433 and 492-495, 1994). Rudinger (in Peptide Hormones, Parsons (ed.), University Park Press: Baltimore, MD, pp. 1-7, 1976).

*Response to arguments*

The applicant argues that the specification provides ample guidance enabling a skilled artisan to make and use the claimed invention without undue experimentation (response, page 6, para.2). The applicant argues that given the teaching of instant specification a person skill in the art can make a vector comprising the SEQ ID NO:1 and 3 and would introduce the vector into pro-nuclei of fertilized eggs of non-human mammals (response, page 6 para.3). Citing various references the applicant concluded that methods of making transgenic mammals other than mice were known in the art at the time of filing (response, page 7 para.1). Thus the present specification fully enables the skill artisan to make and use the claimed invention without undue experimentation.

However, this is found NOT persuasive because applicant's argument alone cannot take place of evidence lacking in the record (see In re Scarbrough 182 USPQ, (CCPA) 1979). The

scope of the claims must bear a reasonable correlation with the scope of enablement (In re Fisher, 166 USPQ 19 24 (CCPA 1970)). At best the specification as filed teaches “a transgenic *Scurfy mouse* whose somatic and germ cells express a transgene comprising a 30kb fragment of normal genomic DNA, including ~7kb coding region of Fkh<sup>sf</sup> gene as well as ~20kb of upstream flanking sequence and ~4kb of down stream sequences that contain a sequence encoding mouse Fkh<sup>sf</sup> protein wherein the expression of exogenous Fkh<sup>sf</sup> transgene results in reduction of T-lymphocyte proliferation in the *scurfy mouse*” (see spec page 33 lines 9-27). Even though applicant argument that method for making transgenic mammals other than mouse is known in the art has been considered the argument is still found unpersuasive, since the invention as claimed is not to a method but to product by process that comprises a transgenic animal with unique phenotype. The state of the art clearly teaches that “The phenotypic changes have been so complicated that they have created more new questions than answers” (see Markkula et al Rev. Reprod. 1996 *ref of record*). The phenotype of an animal is determined by a complex interaction of genetics and environment. The phenotype is influenced by genetic background, which is the collection of all genes present in an organism that influence a trait or traits. The genes may be part of same biochemical or signaling pathway or of an opposing pathway or may appear unrelated to the gene being studied. Furthermore, allelic variations and the interactions between the allelic variants also influence a particular phenotype. These epigenetic effects can dramatically alter the observed phenotype and therefore can influence or alter the conclusions drawn from the transgenic or knockout models. see Wood and Sigmund (*supra*).

Furthermore making transgenic mouse, rat, rabbit, sheep goat or pig wherein the expression of Fkh<sup>sf</sup> transgene is regulated by any transgene construct is not considered routine in

the transgenic art, since gene of interest, promoter, enhancer, coding or non-coding sequences present in the transgene construct and the site of integration, are the important factors that govern the expression of a transgene (*supra*). Furthermore, the specification only teaches a mouse Scurfy model whereas the scope of invention as claimed encompasses a non-Scurfy transgenic animal. Considering the instant disclosure it is unclear whether the over expression Fkh<sup>sf</sup> transgene would result in the reduction of T-lymphocyte proliferation in a non-Scurfy transgenic mouse, rat, rabbit, sheep goat or pig wherein the transgene comprises any mouse or human variants of Fkh<sup>sf</sup> gene.

In addition screening of any and all natural and non-natural variants of mouse and human Fkh<sup>sf</sup> polypeptides (*that modulates T-lymphocyte proliferation*), wherein unknown numbers of amino acid sequences are added substituted and /or deleted when compared to the amino acid sequences of SEQ ID NO:2 and 4 is not considered routine. Making and testing a point mutation is significantly different from the making and testing an amino acid sequences wherein unknown amino acids are added, deleted and/or substituted. The number of possible scenario increase geometrically with increase in percent non-identity. Such making and testing is nothing more than an invitation to further experimentation, since the specification can not be relied on to teach how to make the variants as claimed. One has to engage in extensive making and testing in order to obtain variants that meet the requirements for the claimed Fkh<sup>sf</sup> activity. Accordingly making a transgenic animal (as claimed) which encodes a transgene comparing any and all variants of SEQ ID NO:1 or 3 is highly unpredictable in view of state of art.

This is not considered routine in the art and the experimentation left to those skilled in the art is unnecessarily, and improperly, extensive and undue. See *In re Wands* 858 F.2d 731, 8

USPQ2nd 1400 (Fed. Cir, 1988). It is noted that the unpredictability of a particular area may alone provide reasonable doubt as to the accuracy of the broad statement made in support of enablement of claims. See Ex parte Singh, 17 USPQ2d 1714 (BPAI 1991). Therefore, one skill in the art would have to engage in excessive and undue amount of experimentation to exercise the invention as claimed. The applicant has not presented enablement commensurate in scope with the claims.

***Conclusion***

No claims are allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sumesh Kaushal Ph.D. whose telephone number is 703-305-6838. The examiner can normally be reached on Mon-Fri. from 9AM-5PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Yucel Irem Ph.D. can be reached on 703-305-1998. The fax phone numbers for the organization where this application or proceeding is assigned are 703-308-4242 for regular communications and 703-308-8724 for After Final communications. Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is 703-308-0196.

*S. Kaushal*  
Patent examiner

*Sumesh Kaushal*  
SUMESH KAUSHAL  
PATENT EXAMINER